diseases. But its underlying mechanism needs to be furtherelaborated. The purpuse of this study was to identify the protective and antiapoptotic effects of luteolin on oxidative injury in H9C2 cardiomyocytes and to clarify the underlying mechanism.

METHODS A model of hydrogen peroxide (H_2O_2)-induced H9C2 cells oxidative injury was established in vitro. The changes in cell viability were examined with an MTT assay to determine the available concentration of H_2O_2 and luteolin. 2', 7'-Dichloroflurescin diacetate (DCFH-DA) and flow lytometry were used to detect the effect of luteolin on ROS level and apoptosis degree respectively. We also used Real time fluorescent quantitative PCR to examine the effect of luteolin on the regulation of caspase-3, bcl-2, bax and the ratio of the latter two.

RESULTS We found that incubation with various concentrations of H_2O_2 (0,25,50,100,200) for 1h caused dose-dependent loss of cell viability and 100μM H_2O_2 approximately reduced the cell viability to 50%. Treatment with 10μM luteolin effectively decreased the level of H_2O_2-induced injury. Result of DCFH-DA indicated that 100μM H_2O_2 also increased the ROS level in H9C2 cells, while luteolin obviously reversed this increase. Moreover, the flow cytometry result suggested that luteolin could effectively inhibit apoptosis induced by H_2O_2 in H9C2 cells. PCR results further verified that luteolin downregulated the expression of caspase-3 caused by H_2O_2, and upregulated the ratio of bcl-2 and bax.

CONCLUSIONS Luteolin protects H9C2 cells from H_2O_2-induced oxidative injury by reducing intracellular ROS level and decreasing apoptosis. The protective and antiapoptotic effects of luteolin may be related to its regulation on decreasing caspase-3 and increasing the ratio of bcl-2 and bax.

GW26-e0461

Exosomes secreted from dendritic cells induce angiogenesis by cardiac microvascular endothelial cells after myocardial infarction

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OBJECTIVES It has been reported that the infiltration of dendritic cells (DCs) significantly increased in infarcted myocardium after myocardial infarction (MI) and DCs ablation impaired angiogenesis post-MI in mice. However, the mechanism of how DCs exert effects on MI is still not completely understood. Exosome (EX) has been known as the messenger between cells, this study was aimed to clarify whether EXs derived from DCs induce angiogenesis by cardiac microvascular endothelial cells via paracrine signaling post-MI.

METHODS DCs were derived from mouse bone marrow-derived DCs (BMDCs) and primary cultured rat cardiac microvascular endothelial cells (CMECs) were used to form vasculatures. BMDCs suspensions were incubated with the supernatant of necrotic or normal cultured HL-1 myocardial cells for 24 hrs respectively (as necrosis or control group). EXs were then isolated from the supernatant of BMDCs (DC-Exosomes, DEXs) and identified by electron micrograph and Western blotting using the exosomal marker. DEXs were added to CMECs and the angiogenesis was evaluated by measuring the tube formation and VEGF expression. Finally, the expression profiling of miRNA in splenic DCs of MI mice was analyzed by Affymetrix miRNA 4.0 chip assays and the significantly up-expressed and highly enriched miRNAs were certified both in DCs and DEXs by quantitative RT-PCR.

RESULTS Confocal imaging showed DEXs could be uptake by CMECs. Compared to the control group DEXs, DEXs from necrosis group significantly up-regulated the expression of VEGF in CMECs and enhanced the tube formation by CMECs. Some miRNAs including miR-16-5p, 23a-3p, 150-5p, and 126-3p which are associated with angiogenesis were significantly up-regulated and highly enriched in DEXs from necrosis group compared to those from control group.

CONCLUSIONS These results suggest that exosomal miRNAs especially angiogenetic miRNAs could be secreted from DCs and promote angiogenesis by CMECs post-MI. Our study may present a potent and novel DEXs-based therapeutic approach for MI treatment.

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miR-124 regulation of NFATC1 and atherosclerosis in apolipoprotein E-deficient mice

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OBJECTIVES Atherosclerosis, a chronic inflammatory disease, is the leading cause of death and disability worldwide. Evidence supports a role for microRNAs (miRNAs) in cardiovascular pathophysiology and atherosclerosis development. Herein, we explore the effects of miR-124 on atherosclerosis in apolipoprotein E(-/-) (ApoE-/-) mice.

METHODS A constrictive collar was placed around the right carotid arteries of that were fed a high-fat diet to induce atherosclerotic plaque formation, miR124a-expressing lentiviral vectors (LV) in the presence or absence of recombinant LVTHM-NFATC1 or pGC-FU-NFATC1 was transfected into right carotid plaques respectively.

RESULTS Up to 3-fold downregulation of miR-124 and about 2-fold enrichment of NFATC1 were detected in the models. Consistently, miR-124-expressing resulted in decreased aortic atherosclerosis, impaired pro-inflammatory burden, as evidenced by reduced blood monocytes, endothelial inactivation- and inflammatory markers in aorta, and pro-inflammatory cytokines, chemokines in plasma of ApoE-/- mice compared with the control group. Not surprisingly, silencing NFATC1 mimicked these effects. However, restoration of NFATC1 effectively and consistently attenuated the atherosclerotic suppression phenotypes elicited by the miR-124. Further analysis identified NFATC1 as a direct target of miR-124.

CONCLUSIONS Taken together, the current results reveal, for the first time, a potential molecular regulation of miR124 on NFATC1, offering a possible therapeutic approach for atherosclerosis.

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Changes of plasma angiotensin II and aldosterone in rat model of salt-sensitive hypertensive induced by sensory denervation

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OBJECTIVES Observe the changes of hypertension and plasma angiotensin II and aldosterone in rat model of salt—sensitive hypertensive induced by sensory denervation and to realize the relationship between the hypertension and the angiotensin 11 and aldosterone, therefore explore the mechanism of the hypertension.

METHODS New—born Wistar rats were injected capsaicin(50 mg / kg) hypodermically and the control rats were injected vehicle. After lactation male rats were chosen, divided into four groups and fed the diets with different salt contents respectively for 16 weeks. The tail systolic pressure, plasma concentrations of angiotensin IIand aldosterone in 4W,8W,12W,16w were detected.

RESULTS The control group and the model group's hypertension in 4w 8w 12w 16w were (107±5.9vs141±3.9), (110±4.7vs153±5.2), (111±4.2vs163±4.2), (105±5.5vs177±5.0)mmHg($P>0.05$), The control group and the model group's angiotensin in 4w 8w 12w 16w were 541±12.1vs 250±11.3), (522±10.3vs 318±12.4), (545±11.4 vs 399±17.6), (532±17.1vs 477±15.7)ng/L($P>0.05$), The control group and the model group's aldosterone in 4w 8w 12w 16w were(642±24.1vs256±22.4), (625±23.3vs342±20.6), (645±31.4vs443±28.8), (629±22.7vs490±19.1)ng/L($P>0.05$).

The blood pressure of CON+NS group have no difference with the CAP+NS group. The blood pressure of CAP+HS group in 4 week was higher than other groups($P<0.05$), rising from 4 week to 16 week. Salt loading reduced angiotensin II as well as the aldosterone concentrations in plasma of rats. Compared with control group, the CON+HS group in 4 week have no difference, rising from 8 week. The angiotensin IIand the aldosterone of the CAP+NS group have no difference with the control group, the CON+HS group was lower than control group. CON+HS group rise from 8 week to 12 week.

CONCLUSIONS The establishment of rat model of salt—sensitive hypertensive induced by sensory denervation may be related to renin-angiotensin··aldosterone system.